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pöA longitudinal follow-up study of a type 2 diabetes  
pöfollow-up cohort positive effect on glycaemic control  
changes in medication

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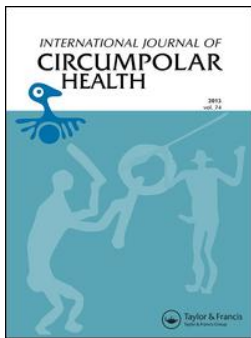
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## A longitudinal follow-up study of a type 2 diabetes “lost to follow-up” cohort – positive effect on glycaemic control after changes in medication

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### ABSTRACT

The aim of this study was to evaluate whether patients with type 2 diabetes (T2D) who had stopped attending their diabetes treatment system (referred to as “lost to follow-up”, LTF) but who succeeded in improving their glycaemic control after returning to the diabetes treatment system had changes in their diabetes medication when compared with similar patients who did not show improvement. “LTFs” who had baseline haemoglobin A<sub>1c</sub> (HbA<sub>1c</sub>)  $\geq 53$  mmol/mol and succeeded in reducing HbA<sub>1c</sub>  $\geq 6$  mmol/mol during a 12–30 month follow-up period after adhering again to their diabetes treatment system were compared with “LTFs” who had an unsatisfactory change in HbA<sub>1c</sub> or with “LTFs” who maintained good glycaemic control throughout the 12–30 month follow-up period. Unsatisfactory change in HbA<sub>1c</sub> was determined as HbA<sub>1c</sub>  $\geq 53$  mmol/mol and change  $< 6$  mmol/mol after the 12–30 month follow-up period in their diabetes treatment system or HbA<sub>1c</sub>  $< 53$  mmol/mol when returning to the diabetes treatment system but  $\geq 53$  mmol/mol at the end of the 12–30 month follow-up period. “LTFs” with improvement in glycaemic control used a higher number of different anti-hyperglycaemic agents ( $P < 0.001$ ) and their dosages of metformin increased ( $P < 0.05$ ) when compared with “LTFs” without improvement or “LTFs” with satisfactory glycaemic control. Cholesterol-, LDL-cholesterol- and triglyceride-concentrations decreased during the 12–30 month follow-up period ( $P < 0.05$ ) in “LTFs” with improved glycaemic control, but not in the other groups. “LTFs” with T2D who had poor glycaemic control seemed to require an increase in their anti-diabetic medication when attempting to improve their glycaemic control.

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Dropout; glycaemic control; medication; primary health care; type 2 diabetes

### Introduction



A proportion of patients with type 2 diabetes (T2D) do not attend diabetes clinics as prescribed by the health-care personnel for a variety of reasons. Poor adherence may be linked to the type of care provided, demographic factors, patients beliefs to medication, and perceived patient burden regarding the treatment procedures [1]. Especially in young patients' inadequate glycaemic control can be explained with poor adherence to medication [2]. These “LTFs” may be exposed to a notable risk of diabetic complications thus reducing their quality of life and raising the costs of diabetes treatment [3–5]. Very little is known about what happens when “LTFs” are recruited back into the T2D treatment system in primary care.

Previous study findings have shown that in the public primary health-care system, 1 in 10 patients with T2D is an “LTF” [4]. Although “LTFs” with T2D are generally

difficult to bring back into the system, those with poor glycaemic control and who are successfully re-attached to the diabetes treatment system seem to benefit from being recalled [5]. Further, this subgroup of “LTFs” with T2D seems to benefit, at least glycaemic control improves, from intensive communication and counselling with the health-care professionals [6]. However, it is unknown whether there are any differences in the changes in diabetes medication between “LTFs” who improved their glycaemic control and those who did not.

Parameters such as blood pressure (BP) and lipid concentrations may also affect, even independently, the wellbeing and cardiovascular outcomes of patients with T2D [7–10]. How these parameters are affected when glycaemic control is improved is unknown.

The aim of the study was to compare the changes made to the diabetes medication in “LTFs” with T2D

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after re-attaching to the diabetes treatment system. As a secondary outcome, we examined whether there were differences in other studied parameters than glycosylated haemoglobin A<sub>1c</sub> (HbA<sub>1c</sub>) between “LTFs” with or without an improvement in glycaemic control.

## Materials and methods

### Study design and setting

This study was a “real-life” longitudinal observational register-based cohort study using data retrospectively obtained from an electronic patient record system. In 2009, a project was initiated for improving the glycaemic control of those who did not attend to T2D care system in the public primary healthcare in the city of Vantaa, Finland. We recorded retrospectively the influence of the clinical work performed by the community primary health-care nurses and general practitioners [4].

The ethics committee of the Hospital District of Helsinki and Uusimaa (Nr 91/13/03/01/2011, 9.1.2013) and the health authority of the city of Vantaa (Dno SOSTER 3124/2011/092) approved this study. According to the ethics committee of the Hospital District of Helsinki and Uusimaa, and health authority of Vantaa, the study participants did not need to sign a Statement of Informed Consent because the study was retrospective, based on patient charts and the investigators did not contact the “LTFs”.

### Study participants

Data collected for the study were based on clinical patient records obtained from the public primary healthcare of the eastern districts of Vantaa, Finland. At the time of the study, Vantaa had a population of 195 397 inhabitants and the eastern districts had 118 802 inhabitants. We identified all patients aged 18–80 years. Patients were determined to have T2D if they had an International Classification of Disease (ICD-10) code E11 in the patient charts or they were prescribed specific anti-hyperglycaemic agents for T2D between 1 January 2005 and 31 December 2009. In Finland, the public primary healthcare used ICD-10 codes to classify diseases, health related disorders, injuries, infections, and symptoms. A computer-based search was made from Finstar (Logica, Helsinki, Finland) patient chart system with a specific report generator.

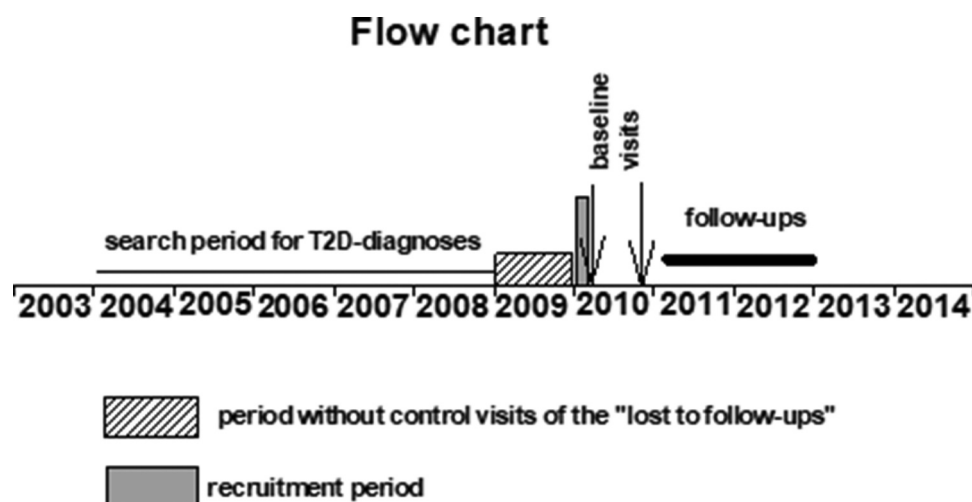
In the eastern districts of Vantaa, 3459 people fulfilled the criteria of having T2D. Patients who fulfilled T2D criteria but had not contacted the public primary health-care system during the previous 12 months

(during 2009) were considered “LTFs” [4]. To establish whether these “LTFs” were true “LTFs” or whether they were receiving treatment elsewhere (e.g. whether they were receiving treatment arranged in another system, private or secondary care), trained diabetes nurses from the public primary health-care system contacted them by phone. If an “LTF” was a true “LTF”, trained diabetes nurses booked an appointment including laboratory tests (baseline visit,  $n = 356$  [4]) in order to improve the diabetes treatment and to bring the “LTF” back to the public primary health-care system. In Finland in that time point, systemic call-back attempts by nurses or diabetes nurses have not been part of the usual public primary healthcare. The follow-up visit was the nearest visit taking place at least one year after the initial visit (12 to 30 months after the baseline visit). Of the contacted true “LTFs”, 32% ( $n = 115$ ) participated both in the baseline visit and the follow-up visit [5] and composed the study population of the study. Figure 1 shows the flowchart of the study.

### Study procedures

At the baseline visit the participant’s status was assessed, blood was drawn and diabetes counselling was provided, and treatment was enhanced when needed [4]. The follow-up visit included the same assessments as the baseline visit. Based on the HbA<sub>1c</sub> measured at the follow-up visit, the success of treatment was evaluated.

“LTFs” were divided into three groups according to their glycaemic control at the baseline visit and at the follow-up visit. One group was defined as “LTFs” with an improvement in glycaemic control, e.g. those who had a HbA<sub>1c</sub> level  $\geq 53$  mmol/mol (7%) at the baseline visit and a reduction in HbA<sub>1c</sub>  $\geq 6$  mmol/mol (0.5%) at the follow-up visit 12–30 months later [6]. The second group was defined as “LTFs” without improvement, e.g. those who had a HbA<sub>1c</sub> level  $\geq 53$  mmol/mol (7%) at the baseline visit and the decrease of HbA<sub>1c</sub> level was  $< 6$  mmol/mol (0.5%) or “LTFs” who had a HbA<sub>1c</sub> level  $< 53$  mmol/mol (7%) at the baseline visit but more than that at the follow-up visit 12–30 months later [6]. The third group was defined as “LTFs” with good glycaemic control, e.g. those who had a HbA<sub>1c</sub> level  $< 53$  mmol/mol (7%) and the HbA<sub>1c</sub> level remained under this value during the 12–30 months follow-up time [4]. According to international guidelines and epidemiological studies [11–14] diabetic complications increase significantly if the HbA<sub>1c</sub> level is  $< 53$  mmol/mol (7%). Therefore, this level was chosen as an indicator of good glycaemic control. The level of change in HbA<sub>1c</sub> (6 mmol/mol, 0.5%) was chosen because



**Figure 1.** Flow chart of the study.

previous studies have shown that reducing HbA<sub>1c</sub> by this amount reduces the incidence of complications in T2D [11,12]. In those patients who returned to the public primary health-care system diabetes treatment was provided according to national and international guidelines, including yearly laboratory, nurse and doctor controls and, if needed, optimising medications [13,14].

### Primary and secondary outcomes

Trained diabetes nurses collected data from the patient records on diabetes medication before and after the 12–30 month follow-up. Based on the medication data, the recruited “LTFs” were divided into two groups: those who were prescribed higher doses of diabetes medication or an increased number of diabetes drugs at the follow-up visit (increased medication), and those who had no changes or who were moved from one oral diabetes medication to another (no change in medication).

As secondary outcomes, we studied patient characteristics including sex, baseline body mass index (BMI), duration of diabetes, diagnosed retinopathy or albuminuria. Baseline BP, blood lipids (cholesterol, low density lipoprotein [LDL] -cholesterol, high density lipoprotein [HDL] -cholesterol, triglycerides), haemoglobin, alanine transaminase, creatinine and prevalence of proteinuria were recorded. When data were available BP, cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides, creatinine and prevalence of proteinuria were recorded and compared with the respective baseline status in all three groups of glycaemic control.

### Statistical analysis

Data are presented as percentage (n) or mean (standard deviation, [SD]) or median (interquartile range [IQR] 25%-75%). Prevalence differences were tested using cross-tabulation and Chi-Square test. Magnitude differences were analysed with Kruskal-Wallis analysis of variance followed by Dunns’ method or ANOVA followed by Bonferroni corrected t-test. Comparisons between before-after situations were carried out with paired t-test, Wilcoxon signed rank-test or cross-tabulation and Chi-Square test when applicable. A P-value <0.05 was considered statically significant.

### Results

#### Characteristics of the study participants at baseline

At the baseline visit, BMI was higher in “LTFs” with improved glycaemic control than in the other groups. At the baseline visit, “LTFs” with improvements had higher triglyceride and lower HDL-cholesterol concentrations than those who maintained good glycaemic control (Table 1). Table 1 shows the characteristics of the study participants at the baseline visit. “LTFs” without improvement had a longer duration of T2D than those who had good glycaemic control (Table 2).

#### Diabetes medication

An increase in dosage or in the number of different T2D drugs during the follow-up was higher in “LTFs” with improvements (32/36, e.g. 89%) than in those without improvement in glycaemic control (17/27, e.g. 63%,  $P < 0.05$ ) or those with good glycaemic control (16/52,

**Table 1.** Characteristics of the study participants (loss of follow-ups, LTFs) at baseline.<sup>a</sup>

Glycaemic control <sup>b</sup>	LTFs with improvement <sup>c</sup> n = 36	Without improvement <sup>b</sup> n = 27	Good control <sup>c</sup> n = 52
Males	16 (44%)	11 (41%)	26 (50%)
Duration of T2D (yrs)	6.5 (IQR25-75%; 3.25–10)	10 (6–10)***	4 (2–8, n = 48)
BMI (kg/m <sup>2</sup> )	33.6 (IQR25-75%;30.6–38.9, n = 29)	30.1 (26.7–32.9, n = 19)*	31.3 (27.2–35.1, n = 39)*
Cholesterol (mmol/l)	4.8 (IQR25-75%;4.2–5.56, n = 34)	4.7 (3.98–5.75, n = 26)	4.8 (3.7–5.6, n = 51)
LDL-cholesterol (mmol/l)	2.8 (IQR25-75%;2.2–3.43, n = 34)	2.8 (2.15–3.33, n = 26)	2.6 (2.0–3.4, n = 51)
HDL-cholesterol (mmol/l)	1.18 (IQR25-75%;0.95–1.33 n = 32)	1.20 (1.10–1.40, n = 26)	1.33 (1.15–1.80, n = 51)*
Triglycerides (mmol/l)	2.2 (IQR25-75%;1.5–3.2, n = 32)	2.1 (1.0–2.5, n = 26)	1.3 (1.1–2.0, n = 51)*
Haemoglobin (g/l)	150.4 (SD:11.5, n = 32)	143.7 (15.3, n = 24)	140.5 (12.8, n = 50)*
Alanine transaminase (U/l)	38.0 (IQR25-75%;25.0–84.5, n = 21)	27.0 (16.3–51.0, n = 12)	28.0 (20.0–33.0, n = 25)
Microalbuminuria (µg/min)	4.5 (IQR25-75%;2.3–25.5, n = 28)	6.0 (2.0–42.5, n = 21)	3.0 (2.0–6.0, n = 47)
Creatinine (µmol/l)	59.0 (IQR25-75%;53.0–72.5, n = 32)	66.0 (62.0–84.0, n = 21)	69.0 (61.0–85.3, n = 48)
Systolic blood pressure (mmHg)	143 (SD:21, n = 30)	149 (22, n = 22)	149 (22, n = 42)
Diastolic blood pressure (mmHg)	90 (SD:11, n = 30)	88 (SD:11, n = 22)	86 (SD:10, n = 42)
Retinopathy	7/27 (26%)	7/23 (30%)	11/35 (31%)
Albuminuria	6/29 (21%)	7/21 (33%)	5/47 (10%)

SD = standard deviation; IQR = inter quartile range; T2D = type-2 diabetes; BMI = body mass index; LDL = low density lipoprotein; HDL = high density lipoprotein.

<sup>a</sup>If results are expressed as median and Inter-Quartile Range25-75% Kruskal–Wallis analysis of variance followed with Dunns' method was used. If results are expressed as mean and Standard Deviation ANOVA followed by Bonferroni method was used. \* stands for P < 0.05 vs. LTFs with improvement Bonferroni method and \*\*\* stands for P < 0.001 vs. good control group, Dunns' method.

<sup>b</sup>Number (n) of available measurements in different glycaemic groups is expressed in the parentheses.

<sup>c</sup>LTFs with improvement: LTFs who had a HbA<sub>1c</sub> level ≥53 mmol/mol (7%) at the baseline visit and a reduction in HbA<sub>1c</sub> ≥ 6 mmol/mol (0.5%) at the follow-up visit 12–30 months later; <sup>b</sup>LTFs without improvement: LTFs who had a HbA<sub>1c</sub> level ≥53 mmol/mol (7%) at the baseline visit and the decrease of HbA<sub>1c</sub> level was <6 mmol/mol (0.5%) or LTFs who had a HbA<sub>1c</sub> level <53 mmol/mol (7%) at the baseline visit but more than that at the follow-up visit 12–30 months later; <sup>c</sup>Good control: LTFs who had a HbA<sub>1c</sub> level <53 mmol/mol (7%) and the HbA<sub>1c</sub> level remained under this value during the 12–30 months follow-up time.

**Table 2.** Number of loss of follow-ups (LTFs) using a specific type of anti-hyperglycaemic agent at the baseline and at the follow-up visit.

LTFs with improvement <sup>a</sup> n = 36	Number of participants at the baseline visit	Number of participants at the follow-up visit
Metformin	24	31
Insulin	7	11
Glitazones	2	4
DPP-4-inhibitors	1	14*
Sulphonylureas	12	7
<b>LTFs without improvement<sup>b</sup></b> <b>n = 27</b>		
Metformin	25	26
Insulin	3	7
Glitazones	7	6
DPP-4-inhibitors	1	7*
Sulphonylureas	7	3
<b>Good control<sup>c</sup></b> <b>n = 52</b>		
Metformin	24	31
Insulin	7	11
Glitazones	2	4
DPP-4-inhibitors	2	12*
Sulphonylureas	12	7

DPP-4 = dipeptidyl peptidase 4.

<sup>a</sup>LTFs with improvement: LTFs who had a HbA<sub>1c</sub> level ≥53 mmol/mol (7%) at the baseline visit and a reduction in HbA<sub>1c</sub> ≥ 6 mmol/mol (0.5%) at the follow-up visit 12–30 months later; <sup>b</sup>LTFs without improvement: LTFs who had a HbA<sub>1c</sub> level ≥53 mmol/mol (7%) at the baseline visit and the decrease of HbA<sub>1c</sub> level was <6 mmol/mol (0.5%) and such LTFs who had a HbA<sub>1c</sub> level <53 mmol/mol (7%) at the baseline visit but more than that at the follow-up visit 12–30 months later; <sup>c</sup>Good control: LTFs who had a HbA<sub>1c</sub> level <53 mmol/mol (7%) and the HbA<sub>1c</sub> level remained under this value during the 12–30 months follow-up time.

\* stands for P < 0.05 vs. baseline status, X<sup>2</sup>-test

e.g. 31%, P < 0.001). “LTFs” without improvement also had more increases in their medication than those with good glycaemic control (P < 0.05). There was an increase in the use of DPP-4 (dipeptidyl peptidase 4)-inhibitors in all studied groups (Table 2). At the baseline

visit, the groups did not differ in the dosage of metformin used. The dosage of metformin increased in “LTFs” with improvements from 1000 mg (median, IQR 25–75%: 0–2500 mg) to 2000 mg (1500–3000 mg, P < 0.05) but not in those without improvement (from



2050 ± 960 mg [mean±SD] to 1956 ± 878 mg) or in those whose glycaemic control remained good (from 1298 ± 1197 mg to 1432 ± 1042 mg). No other systematic changes in medication were observed during the follow-up (Table 2).

### Changes in different parameters during follow-up in different glycaemic control groups

About half (23–30/52) of the “LTFs” who remained in good glycaemic control also underwent other laboratory tests and BP checks than HbA<sub>1c</sub>. Roughly one third of those “LTFs” who were not in optimal glycaemic control at baseline but showed improvement (9–16/36) or who showed no improvement (10–16/27) participated in the T2D check-ups during the follow-up period. In “LTFs” with improvement and who participated in these check-ups, total cholesterol, LDL-

cholesterol and triglyceride concentrations decreased (Table 3).

### Discussion

Improved glycaemic control in recruited “LTFs” was associated with increases in both dosage and the number of drugs used for T2D treatment. There seemed to be changes in medications, especially an increase in the use of DPP4-inhibitors, even among those “LTFs” with good glycaemic control during the follow-up. The laboratory follow-ups were generally performed unsatisfactorily and good clinical care guidelines [13,14] were not followed adequately. A longer duration of T2D was associated with worse glycaemic control. Non-optimal lipid status seemed to improve if a “LTF” was able to improve his/her glycaemic control during follow-up.

Among participants with improvement in glycaemic control, the main change was an increase in the number

**Table 3.** Change in different parameters during the 12–30 month follow-up period in different glycaemic control groups of loss of follow-ups (LTFs).<sup>a</sup>

LTFs with improvement <sup>c</sup> n = 36	At the baseline visit	At the follow-up visit
Weight (n = 9, kg) <sup>b</sup>	100.8 (SD:16.5)	96.1 (16.2)
Cholesterol (n = 12, mmol/l)	5.1 (IQR25–75%:3.8–6.1)	4.4 (3.2–4.9)**
LDL-Cholesterol (n = 12, mmol/l)	3.3 (IQR25–75%:2.0–3.6)	2.6 (1.7–3.1)**
HDL-Cholesterol (n = 11, mmol/l)	1.3 (IQR25–75%:0.8–1.5)	1.3 (1–1.4)
Triglycerides (n = 11, mmol/l)	2.2 (SD:0.8)	1.7 (0.8)*
Microalbuminuria (n = 13, µg/min)	5.0 (IQR25–75%:2.5–29.5)	8.0 (3.0–55.5)
Creatinine (n = 16, µmol/l)	70.2 (SD:20.9)	70.4 mg/l (18.4)
Systolic BP (n = 8, mmHg)	146 (SD:19)	140 (13)
Diastolic BP (n = 8, mmHg)	90 (SD:13)	82 (10)
<b>LTFs without improvement<sup>b</sup></b> <b>n = 27</b>		
Weight (n = 10, kg)	94.0 (SD:17)	93.2 (16.4)
Cholesterol (n = 16, mmol/l)	4.9 (SD:1.3)	4.6 (1.0)
LDL Cholesterol (n = 16 mmol/l)	3 mmol/l (SD:1)	2.8 (0.8)
HDL-Cholesterol (n = 16, mmol/l)	1.1 mmol/l (IQR25–75%:0.9–1.3)	1.1 (0.9–1.3)
Triglycerides (n = 16, mmol/l)	1.9 mmol/l (IQR25–75%:1.3–3)	1.7 (1.3–2.6)
Microalbuminuria (n = 11 µg/min)	4.0 (IQR25–75%:2.0–18.0)	2.0 (2.0–18.0)
Creatinine (n = 14, µmol/l)	65.4 (SD:17.3)	73.2 (18.4)*
Systolic blood pressure (n = 12, mmHg)	137 (SD:22.0)	137 (23)
Diastolic blood pressure (n = 12, mmHg)	88 (SD:11)	81 (10)
<b>Good control<sup>c</sup></b> <b>n = 52</b>		
Weight (n = 23, kg)	97.5 (SD:24.9)	96.8(25.3)
Cholesterol (n = 29, mmol/l)	4.8 (SD:1.0)	4.6 (1.1)
LDL Cholesterol (n = 30, mmol/l)	2.9 (SD:0.9)	2.6 (SD:0.9)
HDL-Cholesterol (n = 30, mmol/l)	1.2 (IQR25–75%:1.1–1.5)	1.2 (1.0–1.5)
Triglycerides (n = 30, mmol/l)	1.8 (IQR25–75%:1.3–2.3)	1.7 (1.1–2.7)
Microalbuminuria (n = 19, µg/min)	6.0 (IQR25–75%:2.0–25.0)	8.0 (3.0–29.0)
Creatinine (n = 27, µmol/l)	67.4 (SD: 17.5)	72.4 (20.8)**
Systolic blood pressure (n = 23, mmHg)	156 (SD 21)	148 (20)
Diastolic blood pressure (n = 23, mmHg)	91 (SD 11)	86 (12)*

SD = standard deviation; IQR = inter quartile range; T2D = type-2 diabetes; BMI = body mass index; LDL = low density lipoprotein; HDL = high density lipoprotein.

<sup>a</sup>If results are expressed as median and Inter-Quartile Range25–75% Wilcoxon signed rank-test was used. If results are expressed as mean and Standard Deviation paired t-test was used. \* stands for p < 0.05 and \*\* for P < 0.01 vs. before status, paired t-test or Wilcoxon signed-rank test.

<sup>b</sup>Number (n) of available measurements in different glycaemic groups is expressed in the parentheses.

<sup>c</sup>LTFs with improvement: LTFs who had a HbA<sub>1c</sub> level ≥53 mmol/mol (7%) at the baseline visit and a reduction in HbA<sub>1c</sub> ≥ 6 mmol/mol (0.5%) at the follow-up visit 12–30 months later; <sup>b</sup>LTFs without improvement: LTFs who had a HbA<sub>1c</sub> level ≥53 mmol/mol (7%) at the baseline visit and the decrease of HbA<sub>1c</sub> level was <6 mmol/mol (0.5%) and such LTFs who had a HbA<sub>1c</sub> level <53 mmol/mol (7%) at the baseline visit but more than that at the follow-up visit 12–30 months later; <sup>c</sup>Good control: LTFs who had a HbA<sub>1c</sub> level <53 mmol/mol (7%) and the HbA<sub>1c</sub> level remained under this value during the 12–30 months follow-up time.

of anti-hyperglycaemic agents prescribed as well as an increase in the dosage of metformin which was not observed in the other two groups. Yet there was no difference in the dosage of metformin between the groups at the beginning of the follow-up and therefore we cannot claim that there would have been a systematic underuse of this drug at the beginning of the follow-up in any of the groups studied. According to international and national diabetes treatment guidelines, which Finnish public primary care is supposed to follow [13,14] metformin is the first-line medication in the treatment of T2D if contraindications do not inhibit its use [15]. Metformin has been shown to improve the prognosis of T2D patients [16]. The increasing communication, and thereby increasing exchange of information, between the T2D treatment system and the recruited “LTFs” with improved glycaemic control which we observed [6] did not solely explain the improved glycaemic status. Recommendation based [13–15] increases in medication also seemed to be required for favourable results in T2D treatment of the “LTFs”.

Interestingly, use of DPP-4-inhibitors increased in all of the groups during the follow-up. Therefore, we cannot conclude that taking these preparations into the treatment repertoire would have had a general positive effect on the glycaemic status of the studied “LTFs”. The DPP-4-inhibitors also belong to the recommended second-line T2D-medications [13–15]. Yet nothing has been shown to be superior to metformin [15,16]. We were not able to show an association between improvement in glycaemic control and an increase in the use of DPP-4-inhibitors so the main reason for the observed increased use of this type of medication may have been aggressive marketing. Just at the time of this study, DPP-4-inhibitors were novel preparations and they were strongly marketed. This may have been the reason for initiating DPP-4-inhibitors as additive medication or replacing parts of previous medication with DPP-4-inhibitors, even in patients with good glycaemic control.

Actions which improved glycaemic control in those recruited “LTFs” who were able to improve their glycaemic control may also have induced modest improvements in some other parameters which are related to better vascular outcomes in T2D patients [8–10]. However, it is difficult to draw very strong conclusions about this because although the general practitioners followed good clinical care guidelines and monitored HbA<sub>1c</sub> concentrations as recommended [13–15] there were other vital parameters which were not as well monitored during the follow-up. Marking T2D diagnosis on patient charts does not guarantee that T2D is properly treated [17] and neglecting actions which belong to proper T2D

treatment is not uncommon in primary care [18–20]. We had difficulties in getting follow-up data about physical measurements such as weight and BP although these parameters may have prognostic value for T2D patients [21]. This holds true also with certain laboratory tests which also may have similar value in the treatment of these patients [21]. Furthermore, the number of eligible patients was small. The present data are 10 years old. According to recent Finnish epidemiological studies [22], the prevalence of T2D has not, however, decreased since the times of these data were collected [23]. Slight changes in recommendations for treatment have been made since that time [13,14]. This could mean that a bigger proportion of putative “LTFs” who finally attend to treatment could nowadays be helped to yield better glycaemic control than was possible in 2010–2012. Yet, this does not rule out the fact that the present data call for better adherence to T2D treatment guidelines in primary care.

## Conclusions

Improved glycaemic control in recruited “LTFs” was associated with increases in metformin dosage and use of other diabetes medications. Improved medication combined with previously described increase in contacting the public primary care that improved glycaemic control may also have induced improvements in some other parameters related to better cardiovascular outcomes in T2D patients.

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## Data statement

The data are available with a reasonable request to the authors.

## Disclosure statement

The authors report no conflict of interest

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